Pathogenesis of *Escherichia coli*:

E. coli **is one of the most common bacteria found in the gut of animals. This includes humans. There are other animal species that contain this organism in the gut including reptiles and fish.** ***E. coli*** **colonizes the gut within a few hours or days of birth depending on the species involved. In humans the intestines can be colonized within 40 hours of birth.** ***E. coli*** **adheres to the mucus overlaying the large intestine. Other bacteria that are growing in the gut may be intestinal pathogens including *Salmonella, Shigella, Yersinia, Enterobacter, Enterobacter, and Klebsiella* all of these may be associated with human illness.**

Physiologically, *E. coli* can grow in the presence or absence of oxygen ($O_{2)}$, this is referred to as being facultatively anaerobic. *E. coli* can respond to environmental signals such as chemicals, pH, temperature, osmolarity, etc., and can swim toward or away from them. In response to changes in the environment *E. coli* can change the size of the outer membrane pores to accommodate larger molecular nutrients or exclude inhibitory substances. Treatment of water with chlorine, bromine or ozone interferes with the membranes ability to transport nutrients.

"*E. coli* is the predominant facultative organism in the human intestinal tract, however, it makes up a very small proportion the total bacterial count. Other bacteria such as *Bacterioides* out number *E. coli* by 20: 1. It is the regular presence of *E. coli* in the intestine that leads to its use as an indicator of fecal contamination of water and wastewater.

Over 700 serotypes of pathogenic *E. coli*, have been recognized based on O(body antigen), H (flagellar antigen), and K (capsular antigen) antigens. Serotyping is important in distinguishing the small number of strains that cause disease. *E. coli* can cause infection in the urinary tract and brain stem (meningitis) as well as intestinal diseases referred to as gastroenteritis.

There are five classes of *E. coli* that produce disease. They are classified by the method of pathogenesis: 1) toxins (enterotoxigenic), 2) invasive (enteroinvasive), 3) hemorrhagic (enterohemorrhagic), 4) pathogenic (enteropathogenic), and 5) aggregative (clumping or enteroaggregative).

The pathogenesis may be described as:

Enterotoxigenic;

These are important in causing diarrhea in infants and travelers especially in regions of poor sanitation. The disease may vary from minor discomfort to a severe cholera like disease. Disease requires colonization and excretion of one or more toxins. These toxins lead to secretion of fluid and resulting diarrhea.

Enteroinvasive;

This type closely resembles shigellosis in its mechanism. These organisms penetrate the cell wall of the colon causing cell destruction and extreme diarrhea. They do not produce shiga toxin.

Enteropathogenic;

The *E. coli* induces a watery diarrhea. The organisms to adhere to the intestinal mucosa and are "moderately-invasive" which causes an inflammatory response. The diarrhea and other symptoms are probably caused by invasion of the host cells and interference with cellular process rather that by production of toxins.

Enteroaggregative;

The distinguishing feature is the ability of the organism to attach to tissue culture cells in an aggregative manner. These strains are associated with persistent diarrhea in young children. They adhere to the intestinal mucosa and cause non-bloody diarrhea and inflamation. This may be related to toxin production.

Enterohemorrhagic (EHEC);

This syndrome is represented by a single strain (serotype O157:H7) which causes diarrhea distinct from some others (including *Shigella*) in that there is copious bloody discharge and no fever. The life threatening situation is its toxic effects on the kidneys (hemolytic uremia). The *E. coli* does not invade the intestinal mucosa as readily as Shigella but does produce two toxins that are virtually identical to shiga toxin. The toxins play a role in the intense inflammatory response and are enhanced by iron deficiency[2] .

At this time *E. coli* O157:H7 is a difficult organism for which to screen. It does not grow well at 44.5°C (defines fecal coliforms from total coliforms) and therefore is not indicated as a fecal coliform. It does not cleave(split) MUG and therefore does not show as *E. coli* in Colilert. It does show as a presumptive positive in P/A broth and as a Total Coliform in Colilert. We should take the Total Coliform and presumptive positives a little more serious than we have in the past.

At this time we sub-culture all presumptive positives in P/A media onto Sorbitol MacConkey's medium which will give us a presumptive positive for *E. coli* O157:H7. The colonies are clear rather than red for the normal E. coli organism. It so happens that Pseudomonas also gives clear colonies and is everywhere in water and makes it difficult to differentiate the organisms. So far, we have been using this procedure for about 2 years on drinking water and have not had an O157:H7 positive verified.

If we did find an isolate of O157:H7 the Diagnostic Lab is able to supply us with anti-sera for serotype identification and Pulse Field gel electrophoresis to determine its exact type.
We are now considering a new medium called CHROMagar O157 but have not put it into production. It is an expensive medium, as all new defined substrate media are, but we will try to move ahead and see if we can obtain some for experimentation.

One more difficulty is, that by the time we finish the analysis, we

would not be able to stop an epidemic. That must be done by the vigilance of the Public Health Offices, City, County Health Departments and especially the physicians and nurses that are attending those individuals reporting with bloody diarrhea.

Selected Bibliography

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